

Conclusions: Exogenous Pim-3 gene can protect rats from LPS/D-GalN-induced fulminant hepatic failure.

PP-032 Hepatoprotective activity of *Momordica cymbalaria* Hook. F against thioacetamide induced hepatic injury in rats

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Objective: The study was aimed at assessing the *in vivo* antioxidant and hepatoprotective activity of methanol extract of fruits of *Momordica cymbalaria* Hook. F. (MEMC) against thioacetamide (100 mg/kg, sc) induced hepatic damage in albino rats.

Methods: The *in vivo* antioxidant activity was determined by estimating the tissue levels of GSH and lipid peroxidation. The degree of hepatoprotection was assessed by estimating levels of biochemical markers like SGPT, SGOT, ALP, bilirubin (total and direct), cholesterol and HDL. 200, 400 and 600mg/kg were used to assess the protective property in thioacetamide model of hepatotoxicity in rats.

Results: The MEMC produced significant effect by decreasing the activity or level of serum enzymes, bilirubin, cholesterol, HDL and tissue lipid peroxidation, while it significantly increased the levels of tissue GSH in a dose dependent manner. The effects of extract were compared with standard, silymarin at 100 mg/kg dose.

Conclusion: These results suggested that methanolic extract of *Momordica cymbalaria* fruits possess hepatoprotective activity against thioacetamide induced hepatic damage and significant antioxidant activity in rats.

PP-033 Preparation and identification of anti-HCMV gBn1 antibody

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Background: Envelope glycoprotein B (gB) of HCMV plays an important role in human cytomegalovirus (HCMV) infection. gBn1 holds an obvious quantitative advantage in HCMV infected Chinese.

Methods: Based on the GenBank sequence (M60929), gBn1 antigen peptide was synthesized. After the gBn1 peptide conjugated Keyhole Limpet Hemocyanin immunized rabbits (including the initial vaccination and three times of enhanced immunization), rabbit antiserum were obtained, then purified by affinity purification. ELISA, western-blot and immunofluorescence methods were used to detect specificity and sensitivity of the antiserum.

Result: Through ELISA, it showed that the antiserum could act with gBn1 peptide, titer could reach 1:64000, and the antiserum could response to the Towne strain, AD169 strain and gBn3 clinical isolate strain. Immunofluorescence methods showed that the antiserum could act with HCMV Towne and AD169 strain. The lysate of HCMV Towne strain, AD169 strain and MRC-5 were as antigen, gBn1 peptide also as antigen, which acted to the antiserum in Western blot analysis. It showed that the serum and Towne stain, AD169 strain, gBn1 peptide appeared a clear band in molecular weight of about 110KD, which was the same as the molecular weight of HCMV gB; no other bands appeared.

Conclusion: By ELISA, immunofluorescence and Western-blot,

the antiserum was able to sensitively and specifically identify HCMV gBn1 peptide. Meanwhile, the antiserum could act with HCMV Towne stain, AD169 stain. It indicated that gBn1 peptide may include epitope. The research on the gBn1 peptide should be carried on, which may help finding the key of HCMV immune mechanism.

PP-034 Protection effect of TanshinonellA against damage in cultured hepatocyte and inhibitory action of TanshinonellA against activated hepatic stellate cell

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Background: To explore the safe dose range of TanshinonellA in hepatocyte and investigate protective effect of TanshinonellA on Tumor necrosis factor α (TNF α) or H₂O₂ damage cell models, and search for inhibitory effect of TanshinonellA upon activated hepatic stellate cell.

Methods: Human hepatocyte line HL-7702 was cultured *in vitro* and treated with different concentrations of TanshinonellA, then cell survival of hepatocyte was detected by MTT assay and supernate ALT and LDH were observed. Establishing hepatocyte models induced TNF α and H₂O₂, supernate ALT and LDH were observed and cell survival of hepatocyte was detected by MTT assay. Rat HSC-T6 was cultured *in vitro* and treated with different concentrations of TanshinonellA, cell survival of HSC was detected by MTT assay.

Result: 1. TanshinonellA has no cytotoxicity to hepatocyte in a certain dose range. Cell survival of hepatocyte decreased and the results of supernate ALT, LDH increased when exceeding the certain dose. 2. TanshinonellA can improve the descent of cell survival of hepatocyte and the increasing of supernate ALT, LDH induced TNF α . 3. TanshinonellA can improve the descent of cell survival of hepatocyte induced H₂O₂. 4. Activated HSC could be inhibited by TanshinonellA.

Conclusion: 1. The safe dose range of TanshinonellA in cultured HL-7702 is 1-2 mg/L. 2. The damage induced TNF α (20 μ g/L) and H₂O₂ (7.5-15mmol/L) could be improved by TanshinonellA in the safe dose range. 3. Activated HSC could be inhibited by TanshinonellA in 25-100 μ g/ml.

PP-035 The protective effect and mechanism of anti-IGFBP-rP1 antibody in the liver tissue of mice with hepatic fibrosis induced thioacetamide

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Background: To investigate effects of anti-IGFBP-rP1 in mice with hepatic fibrosis induced thioacetamide (TAA), and explore if mechanism is relate to TGF- β 1/Smad3 signal path.

Methods: Thirty male Kunming mice were randomly divided into five groups, including control group (A); TAA-four-week adding anti-IGFBP-rP1 treated one week group (B) and TAA-four-week group (C); TAA-five-week adding anti-IGFBP-rP1 treated one week group (D) and TAA-five-week group (E). Hepatic tissues were examined expressions of α -SMA, Collagen I, fibronectin (FN), TGF- β 1 and Smad3 with both immunohistochemistry and Western blot. The apoptosis of hepatic cells was detected by TUNEL.

Result: Contents of both ALT and LDH was significantly increased in C and E. It was significantly decreased in B compared with that of C; also in D compared with in E. Changes of α -SMA, Collagen I, FN, TGF- β 1 and Smad3 in hepatic tissues were significantly increased in C and E. It was significantly decreased in B compared with in C; also in decreased in D compared with in E. Expressions of Smad3 have positive correlation with α -SMA, Collagen I, FN,

TGF- β 1 in each group. The apoptotic rate of hepatic cells in B was significantly reduced than that of C.

Conclusion: Anti-IGFBP-rP1 can prevent the progression of hepatic fibrosis through inhibiting activation of HSC, reducing the expressions of Collagen I and FN, reducing hepatocyte apoptosis and so on. The possible mechanism may relate to the TGF- β 1/Smad3 signal path.

PP-036 Dynamic changes of both IGFBPrP1 and transforming growth factor β 1/Smad on liver tissue with fibrosis in mice

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Background: To investigate dynamic expressions of IGFBPrP1, TGF β 1 and Smad3 of mice with thioacetamide (TAA) - induced hepatic fibrosis.

Methods: Liver fibrosis model of mice was made by intraperitoneal injecting with 5% TAA, 200mg/kg, three times per week, totally for 4 or 5 or 6 weeks. Collagen accumulation in liver tissues was detected by Masson staining. Distribution and dynamic expressions of IGFBPrP1, α -SMA, Collagen I, FN, TGF β 1, Smad3 were detected by immunohistochemistry staining. Meanwhile, expressions of IGFBPrP1, α -SMA, FN and Smad3 were examined by Western Blot.

Result: During the progression of hepatic fibrosis, expressions of Collagen, IGFBPrP1, α -SMA, Collagen I, FN, TGF β 1, Smad3 were gradually increased. Correlation analysis of immunohistochemical staining: during liver fibrosis developing phases, IGFBPrP1 was significantly positively correlated with α -SMA, Collagen I, FN, TGF β 1, Smad3. The results of Western Blot analysis: Molecular weight of β -actin, IGFBPrP1, α -SMA, FN, Smad3 were 43, 31, 45, 220 and 58kD. The contents of IGFBPrP1, α -SMA, FN, Smad3 were significantly increased in model group compared with control group, which were gradually increased with process of fibrosis. There was a significant difference among model group. Correlation analysis of Western Blot analysis: There was a positive correlation between the expression of IGFBPrP1 and the expression of α -SMA, FN, Smad3.

Conclusion: IGFBPrP1 was involved in the formation and development of hepatic fibrosis; Meanwhile, this function of IGFBPrP1 probably relates to promote activation of HSC; promote the synthesis and secretion of both collagen I and FN; affect TGF β 1/Smad3 pathway.

Poster Presentation – Diagnosis & Laboratory Systems Development

PP-037 Screening of specific serum biomarker of ankylosing spondylitis from a random peptide library

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Objective: To screen the specific serum biomarker of ankylosing spondylitis using peptide library biopanning technique.

Methods: Phage random peptide library of 12 amino acids was immunoscreened with purified IgG from sera of ankylosing spondylitis (AS) patients. Positive clones which were obtained after 3 rounds of biopanning were detected by ELISA and 7 of them were sequenced. The binding test of positive clones with the AS patients, systemic lupus erythematosus (SLE) patients, rheumatoid arthritis (RA) patients, Osteoarthritis (OA) patients as well as healthy controls were detected using phage-ELISA. The correlation analyses were evaluated among the absorbance and erythrocyte sedimentation rate (ESR) and C- reactive protein (CRP).

Result: After 3 rounds of screening, the ratio of output to input increase to 1.9×10^{-5} . At the 3rd round of screening, 20 clones were selected and 17 were proved to specifically react with the sera of AS patients. The 7 clones sequenced were come from the same one named AS1. Its inserted sequence was deduced to be QSQRARSIMMM. The positive rate of AS1 in diagnosis AS patients, SLE patients, RA patients, OA patients and healthy control group was 92.0%, 56.7%, 50.0%, 13.3% and 14.0% respectively, and there was significant difference ($\chi^2=77.418$, $P < 0.01$). The absorbance values showed positive correlation with ESR and CRP, the correlation coefficients were 0.165 and 0.239.

Conclusion: These finding indicated that the short peptide QSQRARSIMMM could be the specific serum biomarker of AS and may be used in laboratory test.

PP-038 Dynamic expression of hypoxia inducible factor-1 α during the development of hepatocellular carcinoma and its clinical values

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Objective: To investigate the dynamic expression and alteration of hypoxia inducible factor-1 α (HIF-1 α) and its pathological features in hepatocellular carcinoma (HCC).

Methods: Hepatomas model was induced with 2-FAA on male SD rats for investigating dynamic changes of HIF-1 α . Liver specimen from HCC patients were collected by self-control method. The expression, cellular distribution, and pathological features of HIF-1 α were analyzed by immunohistochemistry.

Results: Rat hepatocytes from granule-like degeneration to atypical hyperplasia and HCC development, and the progressing increasing of the levels of hepatic HIF-1 α and HIF-1 α mRNA expression during the course. The levels of HIF-1 α in hepatoma tissues and sera were significantly higher than those in normal and degeneration ones. There was positive relationship of HIF-1 α levels between them in hepatoma tissues and sera ($P < 0.05$). The positive HIF-1 α as brown and granule-like, mainly presented in cytoplasm and few in nucleus. The incidence was 80% (28/35) in HCC, and 100% (35/35) in its surrounding tissues ($P < 0.001$), respectively. The clinical pathological features of HIF-1 α expression demonstrated that it correlated with tumor size, and its intensity was negative correlated with the differentiation of HCC. No correlation was found between HIF-1 α and tumor numbers or serum AFP level or positive-HBsAg.

Conclusions: Hepatic HIF-1 α overexpression are associated with development and prognosis of HCC.

PP-039 Determine the new serotype 6C Streptococcus pneumoniae by serological method

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Objective: To prepare serotype 6C diagnostic Streptococcus pneumoniae antiserum, to determine it was a new serotype.

Methods: Serotype 6C was determined in USA by using multibean assay. Immunizing rabbits with serotype 6C pneumococcal strains for One month. Detected the capsular titres of antiserum with 6A, 6B and 6C before absorbed and latter by cross-reaction strains.

Results: None capsular reactions were detected from the an-

Table 1. Capsular titers of serotype 6C rabbit antisera to type 6A, 6B and 6C

Antisera	1×	2×	4×	8×	16×	32×	64×	128×
6A	+	+	+	+	+	+	+	—
6B	+	+	+	+	+	+	—	—
6C	+	+	+	+	+	+	+	—